PHARMACOLOGY

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ANS REVIEW LECTURE NOTES

بسم الله الرحمن الرحيم

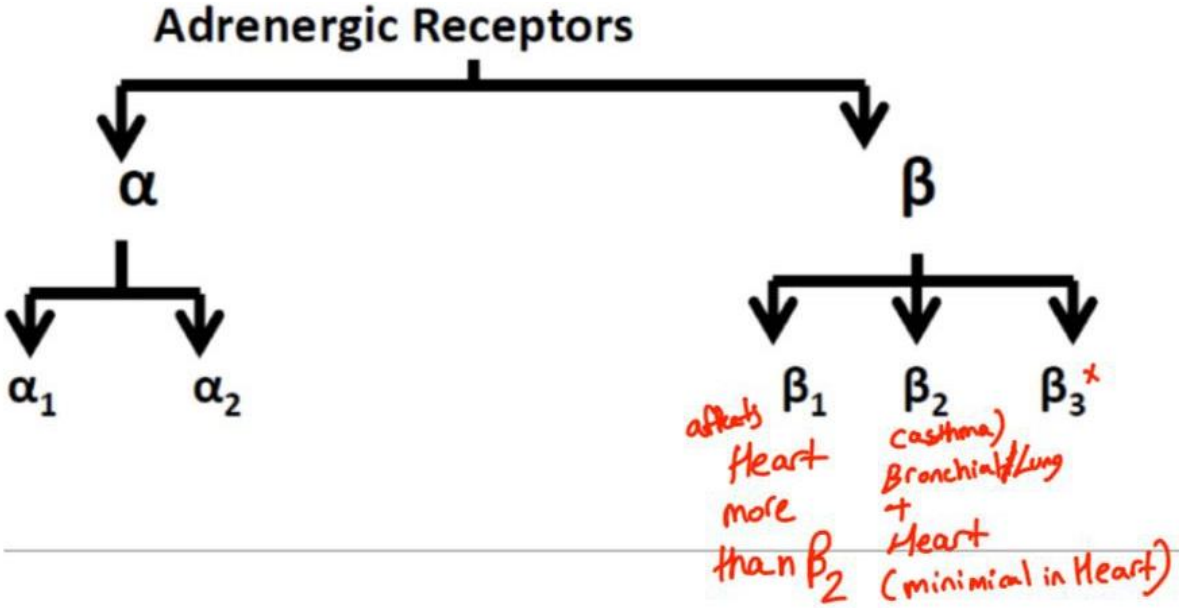
Remember
ANS: Involuntary Movement
Agonist: makes physiology
Antagonist: opposes physiology

- **Neurotramsmitters**: Neurotransmitters are the chemical mediators released by the neurons to transmit the signals through the synapse.
- **Sympathomimetic:** a drug that activates sympathetic nervous system
- **Parasympathomimetic**: a drug that activates parasympathetic nervous system
- **Sympatholytic:** a drug that decreases or blocks sympathetic response
- **Parasympatholytic**: a drug that decreases or blocks parasympathetic response

Sympathetic NS
-fight or flight

-neurotransmitters: Adrenaline/Noradrenaline (the same as epinephrine/norepinephrine) cause increased dilation and heart contraction. On the other hand, they have inhibitory effects on GI, secretions, intestines.

-alpha, beta receptors
In context of asthma, beta agonists are a recommended medication.
In context of hypertension (atherosclerosis), beta blockers are a recommended medication.
But using beta blockers in individuals with both asthma and hypertension is not recommended as that will cause more bronchoconstriction.



Adr:	$\alpha_1 + \alpha_2 + \beta_1 + \beta_2$	adrenaline
NA:	$\alpha_1 + \alpha_2 + \beta_1$ but no β_2 action	Nor adrenaline
Iso:	$\beta_1 + \beta_2$ but no α action	

-Adr/Iso: bronchodilators
-Beta agonists end with ol eg: albuterol
-Beta blockers end with lol eg: atenolol
-alpha blockers end with sin eg: prazosin
-Adr increases heart rate by increasing the automaticity of SA node, cardiac contraction increases.
-Cardioselective beta1 blockers: affect the heart only

Contraindication

- ADR is contraindicated in hypertensive, hyperthyroid, and angina patients
- It should not be given to patients receiving β blockers (a marked rise in BP can occur)

Alpha agonist: vasoconstriction (both alpha 1 & 2)
Beta 2 agonist: vasodilation (skeletal muscle, liver, coronaries)
Beta 2 stimulants for asthma.

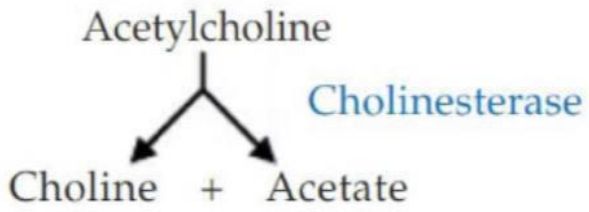
General effects of α blockers

Blockade of vasoconstrictor α1 also α2 receptors ↓ peripheral resistance and causes pooling of blood (Hypovolemia) → ↓ venousreturn and cardiac output → ↓ BP

Alpha blockers have no effect on adrenergically induced cardiac stimulation, bronchodilation because these are predominantly mediated through beta receptors. (They affect vessels and cause vasodilation)

Parasympathetic NS
-rest and digest
- neurotransmitters: Acetylcholine → cholinergic receptors
- Ach has a key role in stimulating GI, secretions, and saliva production.
- cholinergic receptors: M 1,2,3,4,5 they could be muscarinic or nicotinic (Nn, Nm), (In GI, muscles & ganglia)
- Nicotinic receptors have no direct therapeutic use
-nerve gases and insecticides are anti-cholinesterase. (cholinesterase inhibitors)

ACh is hydrolyzed by the enzyme cholinesterase, and choline is recycled immediately after release



Ach contains ester

Cholinoceptors

Two classes of **cholinoceptors** are **muscarinic** and **nicotinic**

Muscarinic

These receptors are selectively stimulated by muscarine and selectively blocked by atropine

They are located in the heart, blood vessels, eye and glands of the gastrointestinal, respiratory, and urinary tracts, sweat glands, and in the CNS

The muscarinic receptors have been divided into 5 subtypes **M1, M2, M3, M4, and M5**

Muscarinic cholinoceptors

The first 3 have been functionally characterized

M1: has a major role in mediating gastric secretion and relaxation of the lower esophageal sphincter caused by vagal stimulation

M2: Cardiac muscarinic receptors are predominantly M2 and mediate vagal bradycardia

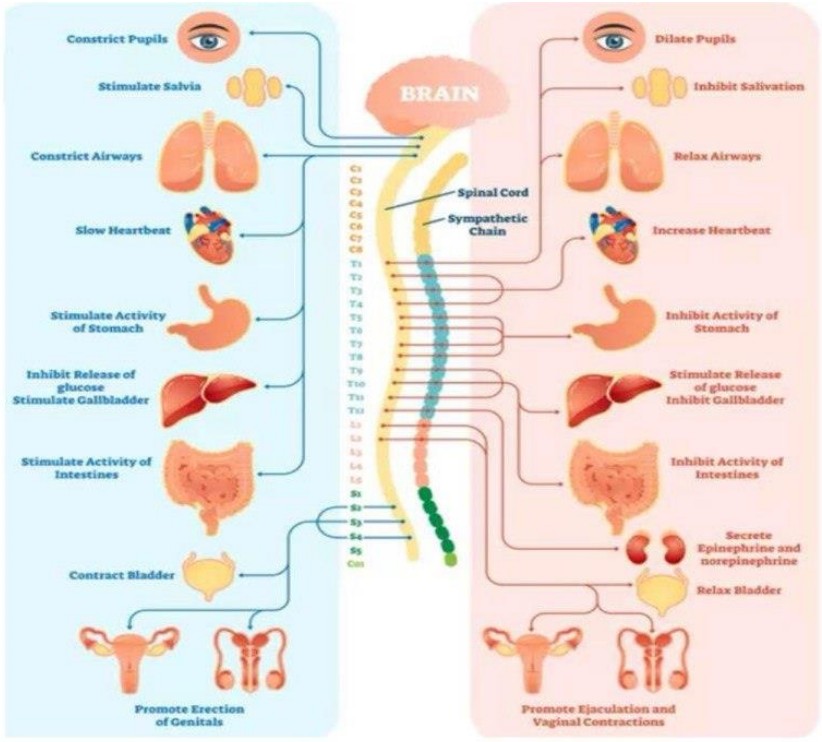
M3: Visceral smooth muscle contraction and glandular secretions are elicited through M3 receptors

Sympathetic: mydriasis (pupil dilation)
Parasympathetic: miosis (pupil contraction)

Cholinergic drugs

They act similarly to ACh, either **directly** by interacting with cholinergic receptors (**agonists**) or **indirectly** by increasing the availability of ACh (**anticholinesterases**)

1. Parkinson (high Ach, low dopamine)
   It is due to dopamine deficiency. //or imbalance between Ach and dop.
   The enzyme that degrades dopamine: monoamino oxidase/catechol-O-methyl transferase… if we give a drug to inhibit these enzymes, dopamine increases, Parkinson level decreases.
2. Cholinergic antidote is atropine (for example prevents salivation)
3. High doses of Ach will have minimal effects on sym NS.



Parasympathetic "Rest and digest" Sympathetic "Fight or Flight"

اللهم سدد رميهم، وثبت أقدامهم، وانصرهم على القوم الظالمين